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PASSIVE SMOKING

P. N. LEE

Independent Consultant in Statistics, 25 Cedar Road, Sutton, Surrey SM2 5DG, England

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Summary—Before 1980 the argument that passive smoking was a serious health hazard was rather tenuous. It was claimed that it produced allergic reactions, impaired driving ability, reduced exercise tolerance in patients with cardiorespiratory disease and increased the risk of bronchitis and pneumonia in first-year children. However, none of these claims provided convincing evidence relevant to the normal healthy adult nonsmoker. Many studies indicate that nonsmokers are unlikely to inhale more than a very small amount of those components of tobacco smoke traditionally considered harmful. It was surprising, therefore, when a study carried out in the USA showed reduced airways function and studies from Japan and Greece showed an increased lung cancer incidence in nonsmokers passively exposed to tobacco smoke in comparison with nonsmokers not so exposed. A review of the detail of these studies suggests that none provides conclusive evidence that passive smoking is seriously harmful, a view supported by a recent large study that was carried out in the USA and in which no significant relationship was found between passive smoking and lung cancer. More research is urgently needed, particularly to explore the influence of potentially confounding factors.

Introduction

Passive smoking is the inhalation of tobacco smoke other than by puffing on a cigarette, cigar or pipe. Study of it is relatively new, with few literature references before 1970. In this review a number of types of accusations that had been levelled against passive smoking up until 1979 are considered first. There follows a section summarizing the dosimetric aspects, understanding of which is fundamental to sensible evaluation of the epidemiological evidence, and then recent suggestions that passive smoking might be a more serious health hazard than hitherto considered likely are examined critically.

Early claims

Annoyance and irritation

That passive smoke exposure, especially under conditions of poor ventilation, can be annoying and irritating is a matter of common experience. By interviewing 250 nonallergic patients about their reaction to cigarette smoke, Speer (1968) found that 69% reported eye irritation, 32% headache, 29% nasal symptoms and 25% cough. Weber, Jermini & Grandjean (1976) found that the frequency of reported eye, nose and throat irritation increased with increasing concentrations of smoke in a sealed chamber and suggested that acrolein was the major offending substance. Subsequently, however, Hugod, Hawkins & Astrup (1978) showed that, although a gas-phase polluted atmosphere was as annoying as one polluted with whole sidestream smoke, air pollution with acrolein at three times the concentration present in sidestream smoke caused considerably less discomfort.

Allergy

The 1979 US Surgeon-General's Report (US Public Health Service, 1979) devoted a chapter to the subject of allergy and tobacco smoke. It concluded that the existence of such an allergy was not clearly established but that those with a history of allergies to other substances, especially those with rhinitis or asthma, were more likely to report the irritating effects of tobacco smoke. Whether this was a psychological, rather than a physiological, response is open to question.

Bronchitis and pneumonia in children

Colley (1974), who has been studying respiratory symptoms in children and young adults for many years, first reported evidence of a possible effect of parental smoking in 1974. In this study a slightly increased prevalence of cough in children aged 6–14 years whose parents smoked lost its significance when parental respiratory disease was taken into account. The author noted that "there was no suggestion that exposure to the cigarette smoke generated when parents smoked had any more than a small effect upon the child's respiratory symptoms".

Later in that year Colley, Holland & Corkhill (1974) published a follow-up paper showing that in children in the first year of life, but not in the second to fifth year, prevalence of cough was significantly higher in children of parents who smoked. This excess was still significant if the analysis was restricted to those parents who did not have phlegm. Despite noting that "the association could be a result of shared genetic susceptibility to respiratory disease between parents and children, to living in the same home environment, and to cross-infection within the family"

Abbreviations: CET = Cigarette equivalent time; COHb = Carboxyhaemoglobin; FEF (25–75%) = forced mid-expiratory flow; FEF (75–85%) = forced end-expiratory flow; FEV = forced expiratory volume in 1 second; FVC = forced vital capacity; NDMA = *N*-nitrosodimethylamine; PM = particulate matter.

they concluded that "a picture has thus emerged of a serious risk to infants in the first year of life from exposure to their parents' cigarette smoke".

A further study by Leeder, Corkhill, Irving *et al.* (1976) sustained these findings, demonstrating an increased prevalence of bronchitis, pneumonia and wheeze (but not asthma) in the first year of life in children whose parents smoked. More possible explanatory factors were studied than in 1974, including whether or not a sibling had symptoms, but standardizing for these did not affect the conclusions.

Whether passive smoke in the atmosphere of the home is the cause of these infections remains open to question. The 1979 US Surgeon-General's Report (US Public Health Service, 1979) suggested that parental neglect may play a role. Also the fact that smokers are more sociable (Eysenck, 1965) could present more opportunity for the children to come into contact with infection.

Psychomotor effects

There has been some concern that relatively low levels of carbon monoxide may have an effect on psychomotor functions, especially in relation to driving a car. The literature reports a great discrepancy in the level at which blood carboxyhaemoglobin (COHb) may affect vigilance. Summarizing the literature, the 1979 US Surgeon-General's Report (US Public Health Service, 1979) concluded that effects seen at levels of COHb found in passive smoking conditions are measurable only at the threshold of stimuli perception and that effects of CO on driving performance and interactive effects of CO and alcohol are only found at higher COHb levels. A recent study by Guillerm, Radziszewski & Caille (1978), in which subjects drove a specially equipped car for 5 hours during the night, exposed either to air or CO sufficient to produce blood levels of 7 or 11% COHb, found no effect of even 11% COHb on driving precision or visual reaction time. This COHb level exceeds that achieved by all passive and indeed most active smokers.

Exercise tolerance

Aronow (1978) examined the effect of passive smoke exposure on 10 patients (two smokers, eight nonsmokers) with *angina pectoris*. Mean time of exercise until onset of angina in control conditions (COHb level 1·3%) was reduced by 22% after exposure to passive smoke in a ventilated room (COHb level 1·8%) and by 38% after exposure in an unventilated room (COHb level 2·3%). He also noted that the passively exposed patients had a raised heart rate and blood pressure. He attributed this to the possible absorption of nicotine, though he did not measure blood levels. The 1979 US Surgeon-General's Report (US Public Health Service, 1979) considered it unlikely that the very low levels of nicotine absorption could be responsible for these physiological changes and suggested that the response could be due to stress following anxiety or aggravation induced by the smoke-filled room.

Summary of evidence available in 1979

Taking all this evidence together, it seemed clear that, while smoking was a source of annoyance to some, although not perhaps very annoying for many, the grounds for believing it to be a health hazard were rather thin. Where adverse effects were claimed they did not apply to the normal healthy adult nonsmoker and/or were not backed by particularly solid evidence. A statement made in a leading article in the *British Medical Journal* (1978) typified the generally accepted view at the time: "For the moment most—but not all—of the pressure for people (including many smokers) to have the right to breathe smoke-free air must be based on aesthetic considerations rather than on known serious risks to health".

Dosimetry

General

A number of totally misleading statements have been made about the dose received by a passive smoker. One example is that by Repace & Lowrey (1980) who, using a theoretical model combined with measurements of cigarette smoke particulate matter (PM) in various different environments, estimated that a nonsmoking office worker exposed to moderate passive smoke inhales the equivalent, in PM terms, of five cigarettes a day while a very heavily exposed nonsmoking musician working in a night club with a chain smoker for a room-mate inhales the equivalent of 27 cigarettes a day. Study of the detail of this paper revealed that the authors had used an extremely low yielding cigarette with a PM yield of only 0·55 mg/cigarette as a basis for calculating cigarettes per day. If, more realistically, a sales weighted average cigarette with a PM yield of 17·6 mg/cigarette had been used, the appropriate exposures would have become one-sixth of a cigarette per day for the officer worker and five-sixths of a cigarette per day for the musician. Even more outlandish was the recently reported claim of Lane quoted in the national press in the UK (*Daily Mail* and *Daily Telegraph* both of 2 June 1981) that "there is now medical evidence to show that the smoke breathed in by non-smokers is 18 times higher in tar and 12 times higher in nicotine than the smoke breathed by smokers ...". The source of this claim undoubtedly comes from a table published by the Laboratory of the Government Chemist (1980), which showed that the ratio of sidestream to mainstream yields was 18 for tar and 12 for nicotine when a very low tar cigarette was smoked under machine conditions. Not only had the cigarette used as a basis for comparison a tar level some 10 to 15 times less than that normally smoked, but the fundamental error of confusing sidestream yields and ambient concentrations had also been made. The concentration of sidestream smoke is measured as it leaves the burning cone of tobacco between puffs, whereas what is relevant to the passive smoker is the concentration of smoke as it reaches him after dilution by room air. Ambient concentrations vary drastically depending on the degree of room ventilation but even under conditions of poor ventilation will be very considerably less than sidestream concentrations, which a nonsmoker would only receive if he were to keep his nose right on top of the cigarette.

A number of workers have measured the concentration of smoke constituents in ambient air and in body fluids. An important study by Hugod *et al.* (1978) measured air concentrations of a number of

Table 1. *Comparison of uptake of smoke constituents in smokers and passive smokers**

Smoke constituent	Mainstream yield inhaled by smoker (mg/cigarette)	Inhaled amount in passive smoking conditions (mg/hr)†	Cigarette equivalents/hr	Cigarette equivalent time (hr)
NO	0·30	0·182	0·61	1·6
CO	18·40	9·160	0·50	2·0
Aldehyde	0·81	0·214	0·26	3·8
Acrolein	0·09	0·013	0·14	7·1
TPM	25·30	2·300	0·09	11·1
Nicotine	2·10	0·041	0·02	50·0
Cyanide	0·25	0·005	0·02	50·0

TPM = Total particulate matter

*Data from Hugod, Hawkins & Astrup (1978).

†Volunteers were exposed in a closed, unventilated room to quite severe passive smoke conditions in which the air CO concentration was kept at 20 ppm over a 3-hr period.

constituents in a closed, unventilated room in which ten volunteers were exposed to quite severe passive smoke exposure conditions in which the air CO concentration was kept at 20 ppm. Comparing the estimated inhaled amounts of each constituent with those inhaled by a smoker they calculated cigarette equivalent times (CET) in hours for seven different constituents (Table 1). It can be seen that these estimates of the time taken to inhale the equivalent of one cigarette vary widely according to the particular constituent.

Nicotine

For nicotine, Hugod *et al.* (1978) estimated it would take a passive smoker 50 hr to take in as much as would a smoker smoking one cigarette, an amount they regarded as negligible. Their results are broadly consistent with those of Hinds & First (1975) who estimated that nicotine concentrations in various public places in the USA ranged from the equivalent of one-thousandth of a filter cigarette per hour in a bus station waiting room up to almost one-hundredth in a cocktail lounge. Similarly Russell & Feyerabend (1975) found that nonsmokers exposed experimentally in an almost intolerably smoky room, whilst having average urinary nicotine levels almost ten times higher than nonsmokers not deliberately exposed to smoke, had urinary nicotine levels 15 times lower than average smokers.

Total particulate matter

For total particulate matter, the constituent usually considered to be related to the excess of lung cancer risk in smokers, Hugod *et al.* (1978) concluded that the CET value is "so high that the passive smoker will never inhale more than what equals ½–1 cigarette per day"—a finding consistent with the conclusions of Repace & Lowrey (1980) if adjusted so that a sensible baseline cigarette is used for comparison.

Carbon monoxide

For CO, the conclusions of Hugod *et al.* (1978) are similar to those of Russell, Cole & Brown (1973) who, working with even more extreme conditions involving twice the exposure level for CO than that used by Hugod *et al.*, found half the CET value (i.e. 1 hour). Even despite this relatively low CET value, it is most unlikely that passive smokers will achieve blood COHb levels as high as 3%, which has been claimed to decrease the threshold for intermittent claudication and angina pectoris in patients with obliterating arterial disease (Anderson, Andelman, Strauch *et al.* 1973; Aronow, Stemmer & Isbell, 1974).

N-Nitrosodimethylamine

N-Nitrosodimethylamine (NDMA) merits mention in the context of passive smoking because of its unusually high ratio of sidestream to mainstream smoke deliveries (Brunnemann, Fink & Moser, 1980) and of its known biological activity.

Brunnemann, Adams, Ho & Hoffmann (1978) measured the levels of NDMA in the atmosphere for several indoor locations in the USA. The highest concentration found (0·24 ng/litre) was in a bar, and the authors calculated that a nonsmoker in this situation would inhale, in 1 hour, an amount of NDMA equivalent to that inhaled by a person actively smoking 17–35 filter cigarettes. Not only was this an extremely smoky atmosphere (their equivalent figure in a bank, where smoking was permitted, being one to two filter cigarettes), but the concentration considerably exceeded that (0·07 ng/litre) found by H. Altmann (personal communication, 1981), in a small (46 m³) unventilated conference room in which 11 people smoked 64 cigarettes in 2 hours—conditions sufficient to produce nausea in the majority of those present. The significance of these low levels of NDMA is not clear. The 1979 US Surgeon-General's Report (US Public Health Service, 1979) points out that the absorption of nitrosamine from environmental conditions is not necessarily equivalent to the absorption by smoking, while Brunnemann *et al.* (1980) have emphasized that "no epidemiological data exist linking human respiratory cancers to volatile nitrosamines".

Dosimetry—a conclusion

Hugod *et al.* (1978) concluded that "in spite of an often considerable subjective discomfort, exposing non-smokers to tobacco smoke under realistic conditions will not cause inhalation of such amounts of the components of tobacco smoke traditionally considered harmful, that a lasting adverse health effect in otherwise healthy, grown-up individuals seems probable".

Table 2. *Vital capacities and expiratory flow rates in smokers and nonsmokers**

Sex	Group number	Smoking habits†	Percentage of predicted			
			FVC	FEV_1	FEF 25–75%	FEF 75–85%
Male	1	Nonsmokers, no smoky environment	102	103	104	120
	2	Nonsmokers, smoky environment	99	98	91	95
	3	Smokers not inhaling	96	99	92	87
	4	Smokers: 1–10 cigarettes/day	95	97	89	77
	5	Smokers: 11–39 cigarettes/day	84	86	76	68
	6	Smokers: >40 cigarettes/day	82	77	72	60
Female	1	Nonsmokers, no smoky environment	102	104	108	112
	2	Nonsmokers, smoky environment	98	99	93	85
	3	Smokers not inhaling	97	99	92	85
	4	Smokers: 1–10 cigarettes/day	96	98	89	83
	5	Smokers: 11–39 cigarettes/day	85	85	78	69
	6	Smokers: >40 cigarettes/day	78	80	72	62

*Data from White & Froeb (1980).
†Exposure to a smoky environment or consumption of cigarettes was for more than 20 yr. Group 3 includes pipe, cigar or cigarette smokers who did not inhale. Groups 4, 5 and 6 were all inhaling cigarette smokers.

Newer evidence

Effects on the small airways

In the last 2 years, some new evidence has caused a considerable amount of rethought on the passive-smoking issue. The first such evidence, published in the *New England Journal of Medicine* in March 1980, came from a study by White & Froeb (1980) of the relationship between various pulmonary function indices and passive smoking. A group of 3002 men and women who had been physiologically evaluated during a "physical fitness profile" course, and who were without a history of relevant cardiorespiratory disease, occupational exposure to dust or fumes or severe exposure to pollution at home or at work were divided into six groups according to their exposure to tobacco smoke. No significant difference was found between nonsmokers exposed to a smoky environment for more than 20 years (group 2) and nonsmokers never so exposed (group 1) as regards forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1); but nonsmokers exposed to passive smoke had statistically significant reductions in forced mid-expiratory flow (FEF 25 to 75%) and in forced end-expiratory flow (FEF 75 to 85%; Table 2).

The most surprising thing about the results of White & Froeb (1980) was that the reductions in FEF seen in group 2 were generally very similar to those seen in group 4, smokers of one to ten cigarettes per day for more than 20 years. Why should a relatively large difference (group 2 *v.* group 1) in airways dysfunction be seen as a result of an apparently relative small difference in exposure to smoke constituents when only a relatively small difference (group 4 *v.* group 2) is seen in response to what was in all probability a much larger difference in exposure?

In view of this implausible result considerable attention had to be given to the details of the study and a number of criticisms were voiced in the *New England Journal of Medicine* (Adlkofer, Scherer & Weimann, 1980; Aviado, 1980; Huber, 1980). One of the oddest things about the study was the procedure by which the sample was selected. It was stated that each candidate was classified into one of the six defined groups. Yet it is clear from Table 2 that anyone who changed smoking habits in the last 20 years does not fit into any group. Furthermore, as the authors define both groups 1 and 2 as living in a house where tobacco smoking was not permitted, what has happened to those nonsmokers, presumably in the great majority, who lived in a house where it was permitted? There are other less important omissions too (inhaling pipe and cigar smokers or cigarette smokers not allowed to smoke at work) and one might even consider it harder to find people who do fit into the groups of White & Froeb (1980) than to find ones who do not. Without an adequate explanation of this anomaly, it is difficult to have much confidence in these findings.

Lung cancer

Whilst the findings of White & Froeb (1980) relate to an index which is contentious and certainly not an accepted reliable indicator of an increased health risk, two more studies published in January 1981, by Hirayama (1981a) and by Trichopoulos, Kalandidi, Sparros & McMahon (1981), caused more attention, as both claimed that nonsmoking wives of smokers had a significantly greater risk of lung cancer than nonsmoking wives of nonsmokers.

Japanese study. Of the two studies, that by Hirayama (1981a), who followed up 91,540 Japanese nonsmoking married women aged 40 years or over in 1965 for 14 years, is the more substantial. He classified women into three groups according to the smoking habits of the husband. The results showed a highly significant trend in the risk of lung cancer with increasing smoking by the husband, with wives of heavy smokers having double the risk of wives of nonsmokers (Table 3). In contrast the wives of smokers had no significant increase in risk for emphysema, asthma, ischaemic heart disease, or cancer of the cervix or stomach.

Following critical comments by Grundmann, Müller & Winter (1981), Kornegay & Kastenbaum (1981), Macdonald (1981), Rutsch (1981) and Sterling (1981), further information on the detail of his study has been given by Hirayama (1981b, 1981c). It is useful to summarize briefly the main points raised and to consider their implications.

Table 3. *Age-occupation standardized lung cancer risk in Japanese women nonsmokers**

Smoking habit of husband	Sample size	Number of lung cancer deaths	Annual lung cancer mortality rate per 100,000	Standardized lung cancer risk ratio	Significance of trend
Nonsmokers or occasional smokers	21,895	32	8.7	1.00	-
Ex-smokers or smokers of 1–19 cigarettes/day	44,184	86	14.0	1.61	$\chi^2 = 10.88$†
Smokers of ≥20 cigarettes/day	25,461	56	18.1	2.08	$P = 0.001$

*Data from Hirayama (1981a).
†One degree of freedom test statistic scoring 0, 1 and 2 for the three smoking categories.

(1) It was suggested that the statistically significant χ^2 value of 10.88 shown in Table 3 might be the result of an arithmetical error as calculations by Mantel (1981), based on the unstandardized data given in the original paper, gave a χ^2 value of only 3.31 which was not significant. In fact the confusion appears to have arisen because Hirayama (1981a) had standardized for age and occupation but had not stated this clearly. This was important as the husbands who smoked were younger than those who did not. In a subsequent letter Hirayama (1981c) presented data by age and occupation which allowed one to calculate a χ^2 value of 8.70 which, while not the same as that originally quoted, was similar and significant. Surprisingly, in the same letter, Hirayama (1981c) quoted a much higher χ^2 value of 36.81 for a similar comparison (difference between nonsmoking women whose husbands have never smoked and those whose husbands have ever smoked) but this was based on a statistical error (Lee, 1981). Despite this error, it seems probable that the association found really was a significant one, though of course a significant association need not imply a significant causal effect of passive smoke exposure.

(2) For a reason that was not apparent, Hirayama (1981a) standardized for age of the husband and not for the age of the wife. However it seems unlikely this would have materially affected the findings, as, if it did cause bias, it would be expected to affect all the causes of death and not just lung cancer.

(3) Smoking habits were determined only at the beginning of the period and may have changed. Again, however, it seems unlikely that enough of these women would have taken up smoking to cause marked bias.

(4) The great majority of the lung cancers seen, 17 out of 23 in a sample, were adenocarcinomas, a type of lung cancer generally believed to be much more weakly related to smoking than squamous cell carcinoma.

(5) Evidence of trends in lung cancer rates in Japan suggest that there may be some other important cause of lung cancer which was not studied. Between 1947 and 1978, female lung cancer rates rose nine-fold and yet Hirayama's (1981a) own results show only a four-fold risk in active cigarette smokers compared with non-passively exposed nonsmokers—and relatively few women in Japan (about 15%) smoke anyway.

(6) The index of "passive" exposure used is not likely to be very accurate. Not only does the husband smoke a varying proportion of his cigarettes at home, but the wife will also be exposed to other sources of exposure besides the husband. In principle, though, this is likely to underestimate rather than overestimate any relative risk associated with passive smoking.

(7) What is most surprising, however, is the sheer magnitude of the association. The two-fold increased risk in wives of heavier smokers is similar, in Hirayama's (1981a) study, to that of women actively smoking about five cigarettes a day, whilst it was stated that the heavy smokers smoked on average only 8.4 cigarettes a day at home and these presumably not all in the direct presence of the wife. If this is so, the study seems to be suggesting that one actively smoked cigarette is not so very different from one passively smoked one, which seems completely inconsistent with the dosimetry, especially when one realises that an active smoker probably has greater passive smoke exposure than a passively exposed nonsmoker.

Greek study. In contrast to the Japanese study, the small Greek case-control study of Trichopoulos *et al.* (1981) is relatively lightweight, being based on only 40 lung cancer cases seen in nonsmoking women. However, their results (Table 4), though having quite wide confidence limits, agree well with those of Hirayama (1981a). Taking into account a number of possible confounding factors (age, duration of marriage, occupation, schooling, residence) did not affect the general picture.

Although the trend is statistically significant, the limitations pointed out by the authors—the small number of cases, 35% of which were not cytologically confirmed, and the cases and controls being taken from different hospitals—would have meant that no great weight would have been attached to the results had they not come out at the same time as, and being supported by, those of Hirayama (1981a). It is interesting, in comparison with the Japanese study, that Trichopoulos *et al.* (1981) specifically excluded adenocarcinomas from their cases, since it was presumably implicitly assumed that this type of lung cancer was not smoking-associated.

American study. Even taken together, the Japanese and Greek studies are by no means totally convincing. Doubts as to whether such a large effect on lung cancer incidence could possibly be due to such an apparently small dose of tobacco smoke have very recently been supported by Garfinkel (1981) based on

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Table 4. *Smoking habits of husbands of Greek nonsmoking women with lung cancer and of nonsmoking control women**

Smoking habit of husband		Lung cancer cases	Controls	Relative risk	Significance of trend
Nonsmokers		11	71	1·0	
Ex-smokers		6	22	1·8	
Smokers (cigarettes/day):	1–10	2	9	2·4	$\chi^2 = 6{\cdot}45$
	11–20	13	32		
	21–30	4	6	3·4	$P < 0{\cdot}02$
	>30	4	9		
	Total...	40	149		

*Data from Trichopoulous, Kalandidi, Sparros & MacMahon (1981).

results from the American Cancer Society's million person prospective study and the US Veterans Study. Two analyses were carried out. The first, similar to that used in the Japanese and Greek studies, showed no significant relationship between lung cancer risk and the smoking habit of the husband (Table 5). Indeed, after matching for age, occupation, education, race, urban/rural residence and absence of serious disease at the start of the study, nonsmoking women married to smokers of 20 or more cigarettes a day had an estimated risk of lung cancer virtually identical to that of non-smoking women married to nonsmokers.

The second analysis found no evidence of any trend in lung cancer rates in nonsmokers over the period of either study. As death rates of smokers had increased substantially over the period, presumably mainly because of the duration of smoking effect (older smokers at the end of the period would on average have smoked for longer than similar aged smokers at the beginning of the period), one might have expected a similar rise to be seen in non-smokers, had passive smoking been a material cause of lung cancer risk in non-smokers.

Although one might argue that passive-smoking effects would be more difficult to pick up in the USA where women spend more time out of the home and marry more often [Garfinkel (1981) had no data on smoking habits of ex-husbands] than is the case in Greece or Japan, it is clear that the Garfinkel (1981) study has underlined the view that further studies are needed to explore the relationship between passive smoking and lung cancer.

Both Garfinkel (1981), and also Hammond & Selikoff (1981) in a paper reviewing findings from the Japanese and Greek studies, pointed out that it is extremely difficult to reconcile findings indicating a higher risk of lung cancer in passive smoking with results from the study by Auerbach, Garfinkel & Hammond (1979) of histological changes in bronchial epithelium taken from autopsy material. Lesions frequently seen in cigarette smokers (such as atypical nuclei and lesions similar to carcinoma *in situ*) have very rarely been found in people who have never smoked. This finding, and also the reported small doses of smoke received by nonsmokers, both suggest that passive smoking cannot play more than a very small role in the development of lung cancer, a view also reached by Lehnert (1981) who considered evidence from the USA and Japanese studies in detail. If passive smoking is not causally implicated it is of fundamental importance to try to identify the confounding or biassing factors that resulted in the higher risk of lung cancer seen in wives of smokers in the Japanese and Greek studies, but not in the American study.

Conclusion

While more research is certainly needed, there seems at present to be no convincing evidence that passive smoking results in any material risk of serious disease for the healthy nonsmoker.

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Table 5. *Lung cancer deaths amongst nonsmoking women in the USA**

Smoking habit of husband	Number of lung cancer deaths	Age standardized analysis mortality ratio	Matched group analysis mortality ratio
Nonsmoker	65	1·00	1·00
Smoker: <20 cigarettes/day	39	1·27	1·37
Smoker: ≥20 cigarettes/day	49	1·10	1·04

*Data from Garfinkel (1981).

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